experiments. Anatomical evidence is at present so scanty regarding afferent nerve fibres from muscle that investigation of their anatomical relation seems absolutely requisite for examining the problem further.

[Just as the lumbo-sacral region of the cord may be split along the median plane without interference to the jerk of either side,* so the same may be done without hindering the above ascending reflex abolition of the jerk. Extinction of the jerk by exciting the central end of the 8th root (from hamstrings) affects the jerk four segments higher without in that distance spreading over to the opposite side. But the excitation affects the jerk of the opposite side if the scope of a considerable length of cord be allowed it. If in the Cat the cord be transversely divided at the 11th thoracic segment, excitation of the afferent fibres from a hamstring muscle of one side (*e.g.*, right) applies chiefly to the jerk on the same side (right), but also to the jerk on the opposite. If, however, in the Cat (in which jerk belongs to the 6th and 5th lumbar segments) the cord be transversely cut at or below the 3rd, the extinction from the hamstring nerve is confined to the same side only. In other words, the presence of additional higher segments seems requisite before the passage of the impulses in question across the median plane of the cord, a fact in curious harmony with an observation by Hallsténţ regarding the elicitation of "crossed reflexes" in the frog. The median posterior column between the 8th and 4th lumbar levels can be removed *in toto* without impairing the influence of the hamstring nerve on the jerk. It is clear also that those fibres of the posterior root which pass to Clarke's column cannot be the requisite afferents, either from the extensor or flexor thigh muscles, because the jerk and the above-described extinction of it are unaffected in the Cat by transverse section of the cord just below the 4th lumbar segment, *i.e.*, the segment where Clarke's column stops short.—*February* 8, 1893.]

III. "On the Leucocytes of Peptone and other Varieties of Liquid Extravascular Blood." By A. E. Wright, M.D., Professor of Pathology, Army Medical School, Netley. Communicated by A. D. Waller, M.D., F.R.S. Received January 30, 1893.

In the course of some investigations on the subject of blood-coagulation, I was led to enumerate the white blood corpuscles in the different varieties of extravascular blood. I propose to report the

* Sherrington, 'Journ. of Physiol.,' vol. 13, p. 666.

† 'Archiv f. Physiol.,' 1885.

results of these enumerations here, and to direct attention to the bearing of the results obtained in the case of peptone blood.

I may premise with respect to the methods employed that the enumerations were made by diluting the blood 200 times with an 8 per cent. solution of magnesium sulphate, which had received a sufficient addition of gentian-violet to stain the cells darkly in the course of a few minutes. The enumerations were invariably made in duplicate samples of each blood, and in each sample the absolute number of leucocytes was counted on 250 squares of the Gowers hæmocytometer. An agreement within 10 per cent. was exacted between the counts of the duplicate samples. When this was obtained the counts were added together, and their sum multiplied by 200 to obtain the total number of leucocytes in the cubic millimetre. The following are the results of my enumerations in oxalated, leech-extract, and peptone blood.

*Oxalated Blood.*

Obtained by drawing off 9 vols. of blood into 1 vol. of a 1 per cent. oxalate of soda solution.

| | Number of white blood corpuscles in normal blood drawn from ear. | Number of white blood corpuscles in blood received from carotid into oxalate solution (figures corrected for dilution effected by the oxalate solution). | Interval between collection of blood into oxalate and commencement of enumeration (*i.e.*, dilution with $MgSO_4$ solution). |
|---|---|---|---|
| Dog 1 ..... | 15,600 | 15,800 | One hour. |
| Dog 2 ..... | 10,400 | 11,200 | Half an hour. |
| Dog 7 ..... | 20,500 | 19,700 | No interval. |
| Dog 8 ..... | 14,000 | 14,600 | Two hours. |
| Dog 14 ..... | 22,300 | 20,600 | One hour. |
| Sum ..... | 82,800 | 81,900 | |
| Average ..... | 16,560 | 16,380 | |

*Leech-extract Blood.*

Obtained by extracting 3 vols. of blood from carotid into 2 vols. of leech-extract. The leech-extract was made from leeches which had been kept under alcohol for several weeks.

| | Number of white blood corpuscles in normal blood drawn from ear. | Number of white blood corpuscles in blood received from carotid into leech extract (figures corrected for dilution effected by leech-extract). | Interval between collection of blood and commencement of enumeration (dilution with $MgSO_4$ solution). |
|---|---|---|---|
| Dog 8 | 14,000 | 13,100 | Nearly two hours. |
| Dog 13 | 16,500 | 14,250 | One hour. |
| Dog 14 | 22,300 | 20 000 | Three-quarters of an hour. |
| Dog 15 | 12,600 | 12,500 | Quarter of an hour. |
| Sum | 65,400 | 59,850 | |
| Average | 16,350 | 14,962 | |

*Peptone Blood.*

Obtained by injection of 0·3 to 0·5 gram per kilo. of body weight of "peptone" in the form of a filtered 10 per cent. solution in 0·75 per cent. of NaCl.

| | Number of white blood corpuscles in normal blood drawn from ear. | Number of white blood corpuscles in peptone blood drawn from carotid. | Interval between peptone injection and blood-letting. | Interval between collection of blood and commencement of enumeration (*i.e.*, dilution with $MgSO_4$). |
|---|---|---|---|---|
| Dog 1 | .. | 2,500 | 10 minutes | One hour. |
| Dog 6 | 30,600 | 3,200 | 8 ,, | ,, |
| Dog 8 | 14,000 | 600 | 5 ,, | ,, |
| Dog 9 | 19,600 | 1,800 | Not noted | Not noted. |
| Dog 10 | .. | 2,800 | 10 minutes | No interval. |
| Dog 14 | 22,300 | 1,300 | 2 ,, | ,, |
| Dog 20 | 14 800 | 1,600 | 5 ,, | ,, |
| Dog 21 | 10,800 | 600 | 10 ,, | ,, |
| Dog 22 | 13,800 | 800 | 25 ,, | ,, |
| Dog 23 | 15,900 | 400 | 10 ,, | ,, |
| Sum | 8)141,800 | 10)15,600 | | |
| Average | 17,725 | 1,560 | | |
| Rabbit 1 | 7,400 | 3,600 | 15 minutes | No interval. |
| | | 750 | 3½ hours | ,, |
| Rabbit 2 | 10,000 | 9,200 | 30 minutes | ,, |
| Rabbit 3 | 8,000 | 4,600 | 2 hours | ,, |
| | | 3,300 | 3 ,, | ,, |
| Rabbit 4 | 8,600 | 4,100 | 30 minutes | ,, |
| | | 2,500 | 3 hours | ,, |
| | | 2,800 | 3½ ,, | ,, |

The figures in column 2 have not been corrected for the dilution effected by the injection of the peptone solution. Taking the blood as 1/13 of the body weight the amount of peptone solution introduced would effect a dilution of from 3·9 to 6·5 per cent. The figures in column 2 ought therefore to be increased by this fraction in order to be strictly comparable with the figures in column 1.

The above tables show that Dog's peptone blood differs from oxalated, leech-extract, and 8 per cent. $MgSO_4$ blood (this last blood was employed throughout as the standard of comparison) in containing a mere tithe of the normal number of leucocytes. These missing leucocytes have either remained behind in the tissues or in the internal vessels, or they have disintegrated and have passed into solution in the plasma. I have endeavoured to decide between these alternatives by making comparative estimations of the leucocytes in the mesenteric veins, and in the carotid blood, and further by making a series of careful histological examinations of the various organs which might be expected to harbour the leucocytes (I selected the liver, the kidney, and the heart muscle for this purpose). In no case was I able to find any trace of stasis or of emigration of leucocytes either in the Dog or in the Rabbit after peptone injections. I, therefore, feel justified in concluding that in all probability the leucocytes have dissolved in the plasma. I believe that this view is borne out also by a consideration of the chemical properties of peptone plasma, notably by the fact that it deposits on cooling a heavy precipitate of a nucleo-albumen, which is probably identical* with Wooldridge's tissue or cell-fibrinogen, in other words, identical with the characteristic albuminous constituent of the white blood corpuscle.

This "cold precipitate" is not obtained from any other plasma except from oxalate plasma, where I have obtained it, after allowing it to stand for 24 hours before separating it from the white blood corpuscles. Under these circumstances a certain disintegration of white blood corpuscles takes place in this plasma. The non-occurrence† of a "cold precipitate" in leech-extract plasma is in accordance with the fact that this plasma contains no disintegrated leucocytes. The non-occurrence of the precipitate in salted plasmas (Halliburton) probably similarly depends on the fact that the white blood corpuscles do not disintegrate readily in these plasmas, but it may be noted that Wooldridge showed that the addition of neutral salts prevented the precipitation of his "cold precipitate."

We have thus reason to believe that the occurrence of a precipitate on cooling peptone plasma is due to the fact that the plasma contains

* *Vide* Pekelharing's identification of Wooldridge's "cold precipitate," or "A-fibrinogen," with the nucleo-albumen of the cell ('Verhandl. d. Konink. Akad. v. Wetenschappen, Amsterdam,' 2nd Sect., Deel 1, No. 3).

† See Dickinson, 'Journ. of Physiol.,' vol. 11.

leucocytes in solution. If this is so, we have a ready explanation of some of the other characteristics of peptone plasma, notably of the fact that the $CO_2$ in this blood is remarkably diminished as compared with the normal blood, and also of the fact that peptone plasma clots when a stream of $CO_2$ is passed through it.

With regard to the diminution of the $CO_2$ in peptone blood, Lahousse (Du Bois-Reymond's 'Archiv,' 1889) surmised that it was due to a driving out of gas from the blood. He based this view on the extreme rapidity with which this diminution occurred in the blood after peptone injection. Blachstein (Du Bois-Reymond's 'Archiv,' 1891), who followed up Lahousse's work, contributed the following to our knowledge of the question. A diminution of $CO_2$ is found in the Rabbit's blood as well as in the Dog's blood after the injection of peptone. In the three experiments reported by Blachstein the $CO_2$ of the normal Rabbit's blood stood to the $CO_2$ of the peptonised Rabbit's blood approximately in the relation of 4 : 3, 4 : 3, and 3 : 2. In his three experiments on Dog's blood the ratios were approximately 3 : 1, 3 : 1, and 2 : 1. It will be noticed that the $CO_2$ undergoes a greater diminution in Dog's peptone blood than it does in Rabbit's peptone blood.

Grandis (Du Bois-Reymond's 'Archiv,' 1891) pursued the subject further, and demonstrated that the tension of $CO_2$ in peptone blood is approximately double that of the $CO_2$ in normal blood. He indicates that the phenomena point clearly to the liberation of some substance with acid properties in the blood.

In view of these facts I would suggest that this substance with acid properties is in all probability the nucleo-albumen of the white blood corpuscles which have become dissolved in the plasma under the influence of the peptone injection. The liberation of this substance in the blood would result in a driving out of $CO_2$ from its combination with the bases of the blood plasma, and would thus account for the great diminution of the $CO_2$ in peptone blood. The differences in this respect between Dog's and Rabbit's peptone blood are in perfect agreement with the results of the enumerations given above for those bloods. The hypothesis of the driving out of $CO_2$ by a liberation of nucleo-albumen in the blood would further harmonise with the increase of the tension of the $CO_2$ in peptone blood, and also with the diminished excretion of $CO_2$ after peptone injections (Bohr, 'Centralblatt f. Physiol.,' 1888).

The fact that casein (better, perhaps, called "caseinogen," Halliburton) will drive out $CO_2$ from $CaCO_3$ constitutes an almost perfect analogy with the property of driving out $CO_2$ which is here surmised to characterise Wooldridge's cell-fibrinogen. In both cases we are dealing with nucleo-albumens.

With respect to the coagulation which is produced in peptone

plasma by passing a stream of $CO_2$* through it, it appears to me that this might be very naturally explained by assuming that we are dealing with a direct reversal of the process which occurs when peptone is injected into the blood. The $CO_2$ coagulation in peptone blood would upon this hypothesis be due to a precipitation of cell-fibrinogen in the plasma under the influence of an excess of weak acid. Such a precipitation of cell-fibrinogen in the plasma would be in some sort an equivalent of an addition of cell-fibrinogen to peptone plasma, and would, therefore, naturally inaugurate coagulation.

The coagulation of peptone plasma *in vitro* by $CO_2$ would be closely paralleled by the fact that intravascular coagulation after injection of cell-fibrinogen occurs, as I have pointed out ('Journ. of Physiol.,' 1890), only in the vascular areas where $CO_2$ is present in excess.

The precipitation of cell-fibrinogen in peptone plasma under the influence of $CO_2$ would further have a close analogy in the precipitation of its congener caseinogen from diluted milk by the addition of excess of dilute acids.

*Presents, February* 9, 1893.

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* This coagulation does not occur in any other plasma; neither does it occur in blood which has been kept liquid by an addition of peptone *in vitro*. It may be noted that the addition of even 8 per cent. of peptone to blood *in vitro* does not entail any destruction of leucocytes.